Advanced Optical Coherence Tomography

Applications for the Retina Specialist

CME Learning Objectives

At the conclusion of this activity, participants should be able to:

• Review advances in diagnostic techniques for retinal disorders that can be performed using available OCT hardware and software.
• Examine the evidence for improved treatment precision using OCT-guided therapy.
• Describe the quantification of retinal pathology obtainable using SD-OCT technology and its clinical interpretation.

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Advanced Optical Coherence Tomography Applications for the Retina Specialist

The essential role of the retina is to observe the outside world; however, until recently, technology for observing the living retina from outside, capable of producing sensitive and detailed anatomical images, was unavailable. This greatly hampered the ability to clearly define, diagnose, and manage the diseases that can affect the retina. Imaging tools, including color fundus photography and fluorescein angiography, allowed many advancements, but could not provide essential details of retinal morphology in health and disease.

The last 15 years have seen marked advancements in retinal imaging, primarily the result of the availability and application of optical coherence tomography (OCT), accompanied by continuous improvements in the scanning speed, sensitivity, and resolution of this modality. Other modalities have not become obsolete, but rather can be used to supplement and, in some cases, remain essential for providing vital diagnostic and management information.

The rapid translation of imaging and image analysis research into the clinic requires practitioners to remain current on the latest technology developments. To provide an up-to-date review of the role OCT can assume in the management of patients with retinal pathology, Vindico Medical Education organized a series of webinars to provide a platform for experts in the field to share their comprehensive experience. The use of OCT imaging to promulgate clear and simple classification of diseases of the vitreomacular interface was described, and specific OCT applications in macular degeneration and macular edema were reviewed.

I thank the panelists for sharing their expertise and participating in the development of this monograph. Readers can expect to enhance their understanding of the role of OCT in imaging the retina and be motivated to ensure they are making the best use of imaging technology that is available to them.

Carmen A. Puliafito, MD, MBA
Course Chair

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Planning Committee and Faculty

Jay S. Duker, MD
Michael S. Ip, MD
Peter K. Kaiser, MD
Carmen A. Puliafito, MD, MBA
Philip J. Rosenfeld, MD, PhD

Medical Writer

Valerie Zimmerman, PhD

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Planning Committee and Faculty members report the following relationship(s):

Jay S. Duker, MD
Stock Shareholder: EyeNefira, Hemera Bioncsiences, Ophthotech, Paloma Pharmaceuticals
Consultant: Akorn/Novartis Pharmaceuticals Corporation, Allergan, EMD Serono, Optos, QLT, Thrombogenics
Research Support: Carl Zeiss, Optovue

Michael S. Ip, MD
Consulting Fee: Allergan, Genentech, Regeneron

Peter K. Kaiser, MD
Receipt of Intellectual Property Rights/Patent Holder: Carl Zeiss Meditec
Consulting Fee: Alcon Laboratories, Inc., ArcitixRX, Bayer, Genentech, Kangshong, Novartis, Ophthotech, Oraya, Regeneron, Thrombogenics

Carmen A. Puliafito, MD, MBA
Consulting Fee: Alcon Laboratories, Inc., ArcitixRX, Bayer, Genentech, Kangshong, Novartis, Ophthotech, Oraya, Regeneron, Thrombogenics

Philip J. Rosenfeld, MD, PhD
Stock Shareholder: DigiSite
Consultant/Advisor: Accella, Alcon, Boehringer Ingelheim, Chengdu Kanghong Biotech, Oraya
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OCT in the Assessment and Management of Disorders of the Vitreomacular Interface

Jay S. Duker, MD

The vitreomacular interface (VMI) is a complex structure linking the vitreous humor, the largest structure in the human eye, with the retina; however, until recently, a reliable method for visualizing and evaluating the VMI in vivo was unavailable. Four pathologies involve this structure: vitreomacular traction (VMT), full-thickness macular hole (FTMH), lamellar macular hole (LMH), and epiretinal membrane (ERM). Their early diagnosis is essential to initiation of treatment when therapy may be most effective for improving visual outcomes. In 2014, spectral-domain optical coherence tomography (SD-OCT) is the critical imaging modality to diagnose and manage these conditions.¹

Anatomy and Composition of the Vitreous

The vitreous is a gel made up of approximately 99% water.² The normal adult vitreous is avascular,³ with mononuclear hyalocytes widely scattered in the vitreous cortex.³ Vitreous macromolecular constituents include collagen fibrils with a scaffold-like structure that is supported by glycosaminoglycans (GAG). These fibrils are interspersed with “inflating” hyaluronan molecules, which comprise 90% of the vitreous GAG content.⁴ Chondroitin sulfate, contributing 10% of GAG content, provides necessary spacing between collagen fibrils, allowing vitreous transparency.

The organization of its structural components provides the vitreous with unique optical and mechanical properties, including stability and minimization of light scattering prior to the age-related development of posterior vitreous detachment (PVD). Fibrils originating from the vitreous base splay out, inserting anteriorly and posteriorly to the ora serrata.⁶ The vitreous is firmly attached at the vitreous base, optic disc, blood vessels, and macula.⁴,⁶

PVD is a long-term, chronic process. Most eyes undergo the normal PVD of aging, occurring in 4 stages, without culminating in macular pathology (Figure 1, page 4). Stage 1 is perifoveolar, progressing to stage 2 with the vitreous elevated from the macula but still attached elsewhere.⁴ During stage 3 the vitreous remains attached at the disk, and a full PVD with Weiss ring characterizes stage 4.

PVD typically occurs gradually over years with progressive liquefaction of the vitreous gel and weakening of the vitreoretinal adhesion.⁷ Areas of liquefied vitreous are observed in eyes as young as 4 years, with the liquid proportion increasing to approximately 20% of the vitreous volume by the late teens.⁴,⁶,⁸ After age 40, liquefaction is progressive, and by 70 years more than half of the vitreous is liquefied. However, PVD is rarely observed in normal eyes at autopsy in those younger than 60 years.⁹ After age 60, liquefaction and PVD are significantly correlated, when the vitreoretinal adhesion becomes sufficiently weakened to allow separation. As aging continues, lacunae of liquefied vitreous increase in number, size, and coalescence, which contributes...
VMI Disorders

A minority of eyes undergo anomalous PVD, which can lead to VMI disorders. Interruption of the normal PVD process can occur through 2 pathophysiologic mechanisms (Figure 2), and several retinal changes can result. Possible ramifications include retinal distortion and thickening; accumulation of subretinal fluid; formation of intraretinal cysts; full-thickness or lamellar macular hole; and schisis, or splitting, of the inner and outer retinal layers.

Persistent vitreous traction on the fovea or optic nerve can occur when an imbalance exists between the amount of liquefaction and weakening of the vitreoretinal adhesion. VMT and FTMH are typical sequelae. Epiretinal avascular proliferation of fibrous tissue can also interrupt normal PVD. Vitreoschisis, when residual vitreous cortex remains on the ILM after PVD, can serve as a nidus for both ERM proliferation and LMH.10

Pathology typically develops where the vitreous is most firmly attached to the retina: at the vitreous base, along large retinal vessels, at the optic disc margin, and at 2 macular locations—a 500-μm radius foveolar attachment and a 1500-μm radius tight foveal attachment.4 When conditions such as myopia, trauma, surgery, and inflammation induce acute PVD, adhesion at the vitreous base can produce a peripheral retinal tear. Foveal attachment size is related to subsequent pathology. Small (<500 μm) focal vitreous attachments can produce tractional changes on the fovea, resulting in VMT, cystoid spaces, subretinal fluid, FTMH, or LMH. Broad (>1500 μm) attachments, which produce lower tractional forces, are less likely to cause macular dehiscence, but are associated with more diffuse pathology, including ERM, traction macular detachment, macular schisis, and hyaloideal thickening in diabetes. These changes may be related to other macular conditions, including diabetic macular edema, wet macular degeneration, and retinal vein occlusion. Some eyes have elements of both of these processes.

OCT for Evaluation of the VMI

The advent of OCT has greatly expanded the ability to observe normal eyes and pathologic changes that may affect them. This widely available, noninvasive technology has assumed a critical role in diagnosis and management of VMI conditions. OCT provides a high-quality, high-resolution, cross-sectional analysis of the macula, producing highly reproducible quantitative evaluations of macular thickness, and also allows imaging of clinically invisible structures, including the posterior hyaloid, vitreous adhesions, and small intraretinal cysts and holes.

Newer systems use SD-OCT (a form of Fourier domain OCT) technology, which is currently the most clinically useful. SD-OCT is much faster than previous time-domain OCT devices. Each A-scan is captured instantaneously, with a spectrometer and a charge-coupled device camera detecting all light echoes simultaneously. The increased speed makes 3-dimensional scanning possible, with precise registration from visit to visit. Faster scanning of the macula leaves no clinically relevant skip areas, and the inner retinal
surface can be better visualized. SD-OCT allows improved delineation of fine vitreous structures, ERMs, and tiny retinal cysts and schisis cavities. These images can confirm FTMH vs LMH and assist with surgical planning.

OCT is an excellent device to assess macular structure. Surgery for macular conditions does not always provide congruent anatomical and visual outcomes; however, some OCT applications can provide a valuable bridge between structure and function. For example, the improved visualization has clarified associations between anatomical status and visual outcomes following surgical repair of macular holes. Disruption of the inner segment-outer segment (IS-OS) junction is associated with poorer postoperative best-corrected visual acuity (BCVA), and OCT demonstrated that BCVA was significantly worse in patients who showed both IS-OS and external limiting membrane disruption.

**Practical Considerations**

- The highest quality line scan (B-scan) should be used for imaging. On the Cirrus HD-OCT 5 line raster, oversampling a single-line scan 20 times is preferred. Using the enhanced depth imaging option slightly diminishes vitreous scan quality. Examination of the entire cube scan is useful for distinguishing an FTMH from an LMH and when evaluating areas of nonfoveal traction and traction at the optic disc.

**OCT-Based Classification of VMI Conditions**

In 2012, an international team of experts developed an anatomic classification of VMI conditions based on OCT without use of clinical signs or symptoms. The system was designed for use in clinical practice and research and to be predictive of surgical outcomes. The classification describes 1 finding (vitreomacular adhesion [VMA]) and the 4 VMI pathologies: VMT, FTMH, LMH, and ERM (Figure 3).

**Vitreomacular Adhesion**

VMA is characterized by detachment of the posterior vitreous cortex (posterior hyaloid) from the retinal surface, outside the foveolar area. Macular attachment of the vitreous cortex must be within 3 mm of the fovea. An essential component is lack of anatomical retinal changes. This finding has 2 subclassifications. The first is based on size of the adhesion, either focal (≤1500 μm) or broad (>1,500 μm). The latter are roughly parallel to the retinal pigment epithelium and may include focal areas of cortex dehiscence.

The second VMA subclassification is based on the presence or absence of concurrent macular disease: isolated (with no other macular disease) or concurrent (other macular disease present). Cause-and-effect relationships between VMA and concurrent disease are currently unproven. Visual and anatomical observations may be due to the VMA or the concurrent disease. Studies are underway to investigate whether release of the VMA can contribute to a better outcome in eyes with a concurrent disease that is associated with macular pathology.

VMA is exclusively an OCT finding that cannot be seen clinically, has no symptoms, is equivalent to stage 1 PVD, and is the result of age-related changes of the vitreous. The condition is extremely common, rarely pathologic, and not necessarily predictive of subsequent pathology.

**Vitreomacular Traction**

VMT has the vitreous features of VMA but is accompanied by abnormal retinal architectural changes on OCT. Per the new classification, “symptomatic VMA” is a clinical diagnosis that requires 2 elements: VMT on OCT and patient-reported symptoms attributed to the retinal anatomical changes. VMT includes detachment of the posterior vitreous cortex from the retinal surface. The detached hyaloid may or may not be thickened. VMT also features macular attachment of the vitreous cortex within 3 mm of the fovea. This condition is always pathologic, but may not be symptomatic.

Similar to VMA, VMT can also be subclassified based on size of the adhesion and presence of other macular disease. As with concurrent VMA, in concurrent VMT anatomical changes, such as persistent cysts within the retina of a patient with wet age-related macular degeneration, may be the result of the VMT or the concurrent disease.

**Full-Thickness Macular Hole**

OCT has allowed remarkable advancement in understanding the pathogenesis, diagnosis, and management of
FTMH, which is characterized by a full-thickness retinal defect involving the fovea. The condition can be subclassified according to size of the defect (aperture size), presence or absence of VMT, and whether the cause is primary (initiated by VMT) or secondary (directly due to associated disease or trauma known to cause macular hole in the absence of prior VMT). This is not a staging system but rather a descriptive system based on OCT findings alone. Macular hole aperture size is divided into small (<250 μm), medium (250 to 400 μm), or large (>400 μm) and is measured at the narrowest width, not at the ILM.

A primary FTMH results from VMA progressing to VMT due to anomalous PVD. During this process, VMT can initially affect the inner or outer retina; which location is more likely to progress to FTMH is unknown. Although the term “idiopathic macular hole” is sometimes used, this phrase should now be considered a misnomer, as the origin of the hole is known. A secondary FTMH is the result of other conditions and does not have preexisting or concurrent VMT. Blunt trauma, high myopia, macular edema, macular schisis, ERM, choroidal neovascularization, and surgical trauma are common precipitating events (Case 4, page 9).

The Gass classification divided macular holes into 4 stages (Table). In the new classification, the previously named stage 0 macular hole is now referred to as VMA in the contralateral eye of a person with existing FTMH, and what previously was referred to as stage 1 macular hole is isolated focal VMT (Case 2, page 9).

OCT has helped to clarify the need for face-down positioning after macular hole repair. This practice was maintained for 1 week postoperatively in the seminal 1991 report in which investigators reported a 42% (22/52) success rate for improved visual acuity. Others have advocated up to 6 weeks of this positioning, which is a significant burden on patients and may eliminate surgery as an option for some. Subsequent studies have shown macular hole closure rates of approximately 90% with little or no face-down positioning. Although current practices include variable lengths of face-down positioning postoperatively, with SD-OCT the macula can be imaged through the gas bubble immediately following surgery. If the hole is closed, theoretically no further face-down positioning may be necessary.

### Lamellar Macular Hole

Gass originally described an LMH as an aborted macular hole, where the inner retina was pulled off during the occurrence of a PVD. Based on SD-OCT, the definition can be expanded as the loss of inner retinal tissue with the outer retina (photoreceptors) intact. A similar appearance has been observed with other etiologies; resolution of VMT, concurrent ERM, cystoid macular edema, and high myopia. Surgery is controversial in these patients and must be reserved for select cases.

### Therapeutic Options

Currently 3 management choices are available for VMT and FTMH. Watch and wait (observation) is supplemented by vitrectomy or pharmacologic therapy with ocriplasmin.

**VMT**

Observation is a reasonable approach to VMT, especially when the condition is of recent onset, visual acuity is good, and symptoms are minimal. However, if the fellow eye has a VMI pathology, the probability for progression in the VMT eye is greater. After 2 years, approximately one-third of eyes with VMT will progress into full PVD, and most of these eyes will have normalized anatomy. Several studies have shown that vitrectomy is beneficial for VMT, with postoperative visual acuity improving by 10 to 15 letters.

**FTMH**

Observation is rarely recommended for FTMH, where natural history indicates that almost all cases will result in a central scotoma with visual acuity at the level of legal blindness. Only small primary FTMH (<250-μm aperture size) of recent onset or associated with high myopia, and traumatic secondary FTMH should be considered for observation. Some of these holes will close spontaneously, and diligent follow-up with OCT imaging facilitates management. If the hole enlarges, vision worsens, or the patient becomes increasingly symptomatic, intervention may be warranted.

Vitrectomy usually produces excellent anatomical and
visual results in patients with FTMH. Outcomes in several studies showed postoperative visual acuity improvement of 15 to 25 letters. However, some constraints exist. A phakic eye usually develops a cataract, some face-down positioning remains the standard of care, and risk of a significant complication is approximately 1% to 2%.18-21

Pharmacologic intervention is another option. Ocriplasmin was introduced in the United States in 2013 for treating VMT (“symptomatic VMA”) and FTMH with VMT.22 This agent is a first-in-class recombinant truncated form of plasmin that targets fibronectin, laminin, and collagen, the molecules that link the vitreous to the ILM, producing a clean separation.23 Ocriplasmin induces both liquefaction and vitreous detachement. Treatment benefits include the convenience of an office-based intravitreal injection, which is less invasive than surgery. Cataract formation is not expected. However, comparative complications between injections and surgery will require more data.

Pivotal phase 3 trials designated the OCT-based structural outcome of VMA resolution at day 28 as the primary endpoint, which is unique for studies typically required to have functional endpoints, such as visual acuity.24 Resolution of VMA occurred in 26.5% of eyes in the ocriplasmin group compared with 10.1% in the placebo group (P<0.001). All ocriplasmin responses occurred by day 28; continued follow-up revealed no additional resolutions. FTMH closure at day 28, a secondary pivotal trial endpoint, occurred in 40.6% of patients in the ocriplasmin group compared with 10.6% in the placebo group (P<0.001).24 The subgroup with small aperture size had 58.3% closure with ocriplasmin compared with 16.0% with placebo; closure of medium-sized holes was 36.8% compared with 5.3%.24 Large macular holes (>400 μm) were excluded from the study; however, some were inadvertently enrolled and treated. None of these eyes had successful FTMH closure (Case 3, page 8).

Patients who did not have FTMH closure during the trials underwent vitrectomy after study closeout. Success rates for macular hole closure were similar between patients in the ocriplasmin group (93.1%) and those who received vehicle (92.3%),25 which suggests that treatment with ocriplasmin does not affect the ability to subsequently close a macular hole with vitrectomy (Case 1, page 8).

Ocriplasmin Pearls

Ocriplasmin therapy must be based on a current OCT image, preferably repeated on the day of injection. VMT must be present; otherwise, ocriplasmin should not be used. The size of the adhesion is important, with those <1500 μm having better outcomes. The size of the hole is also important, with closure of almost two-thirds of holes <250 μm, decreasing to approximately one-third of holes between 250 and 400 μm. Ocriplasmin should not be used if the hole is >400 μm. Presence of ERM is a relative contraindication, as positive results are rarely achieved with concomitant prominent ERM. Patients should be counseled to expect symptoms of a PVD immediately after a successful injection. If the injection is unsuccessful, nothing indicates that ocriplasmin treatment will affect vitrectomy success.

Summary

Pathologies of the VMI are caused by anomalous PVD. OCT is the critical tool for managing these conditions. Observation is indicated for mild to moderate cases of VMT but is reserved for select cases of FTMH. Vitrectomy has a high success rate for anatomical and visual improvement in both groups. Ocriplasmin is the first FDA-approved pharmacologic treatment for “symptomatic VMA,” that is, VMT and FTMH with VMT. Case selection is critical with all therapies.

References


Case 1: Secondary Full-Thickness Macular Hole
A 49-year-old woman with high myopia presented with recent onset central distortion in her left eye and 20/50 vision. Visual acuity was 20/20 in her right eye. SD-OCT did not show VMA or VMT but revealed an early schisis in the fovea (Figure 1A). The patient returned after 2 weeks reporting a sudden worsening of her vision, which was now 20/60. An FTMH was evident, and she continued to have no VMA or VMT (Figure 1B). She was treated with vitrectomy and gas, and her vision was 20/20 at her 1-month follow-up (Figure 1C).

Case 2: Importance of Contralateral VMT
A 57-year-old woman presented with an FTMH in the left eye (VA 20/200) and a normal right eye (VA 20/30), with no sign of VMA or VMT (Figure 2A). Six months after successful vitrectomy in the left eye, vision in the contralateral eye remained 20/30, and the patient was asymptomatic. However, broad, isolated VMA (stage 1 PVD) was evident in the right eye and, after another 6 months, became isolated focal VMA. Two years after the vitrectomy in the left eye, the patient was symptomatic and had isolated focal VMT in the right eye, although her visual acuity remained 20/30. Four years after the vitrectomy, she continued with focal, isolated VMT (Figure 2B) and had increasing symptoms, including metamorphopsia, with 20/30 vision. Five years after her initial presentation, VA in her right eye was 20/80. A posterior vitreous detachment was observed in the right eye, and despite VMA/VMT release, a small, primary FTMH occurred, with release of VMT (Figure 2C). Vitrectomy was
performed, and 1 week postoperative the hole was closed, and her vision was 20/50 (Figure 2D).

**Case 3: Lamellar Hole with Moderate Myopia**

A 53-year-old man with moderate myopia with 20/40 vision and mild distortion was shown to have an epiretinal membrane and a lamellar macular hole (Figure 3A). After observation for 1 year, his vision deteriorated to 20/60, and he was increasingly symptomatic (Figure 3B). He underwent vitrectomy and ERM and ILM peel. Although not an FTMH, gas was injected. The patient had a good anatomical and visual outcome (20/40) at 1 month postsurgery (Figure 3C).

**Case 4: Small Macular Hole Repair**

A patient had a small, primary macular hole in the right eye with release of VMT (Figure 4A). His vision was 20/50 in that eye. He underwent surgery with peeling of the posterior hyaloid and SF6 gas bubble. OCT on day 1 postsurgery indicated the hole was closed (Figure 4B). The patient did 3 additional days of face-down positioning, with successful hole closure and vision of 20/20 at 1 year (Figure 4C).
Age-related macular degeneration (AMD) is the leading cause of blindness in older adults in developed countries.1 AMD has several well-defined stages based on the appearance of the macula. Early and intermediate stages are characterized by drusen and pigmentary changes, while geographic atrophy (GA) and neovascularization are typical of late-stage AMD.2 Historically, color fundus, autofluorescence, and reflectance imaging were used to follow AMD. However, optical coherence tomography (OCT) has recently allowed detailed investigation of structural changes that occur during disease progression, and this modality has become the gold standard for monitoring therapy.

**AMD and Anti-VEGF Therapy**

Neovascular AMD is primarily the response to excess vascular endothelial growth factor (VEGF) in the macula, which OCT identifies by the presence of fluid in and under the retina and under the retinal pigment epithelium (RPE). Several strategies have evolved for managing neovascular AMD with anti-VEGF therapy: OCT-guided as-needed (PRN) dosing, treat-and-extend dosing, and monthly or bimonthly dosing without OCT monitoring. OCT-guided treatment is possible when response to anti-VEGF injections, characterized by a reduction of macular fluid, is followed on OCT retinal-thickness maps and individual B-scans.

**SD-OCT Technology**

Custom automated algorithms for specific structure features of the macula enhance spectral-domain OCT (SD-OCT) capability. Processing that produces a structural image from the recorded signal proceeds through several steps that include resampling and background subtraction. The resulting *en face* image is restricted to the scanned area. SD-OCT imaging of vascularized pigment epithelial detachments (PEDs) is based on the algorithms developed for drusen imaging that easily and reproducibly measure drusen volume and area. Algorithms vary in availability and characteristics among SD-OCT instruments.

In the Cirrus HD-OCT 200 x 200 raster scan pattern, each B-scan has 200 A-scans, and each of the 6 mm × 6 mm raster scan patterns has 200 B-scans. The A- and B-scans are separated by 30 μm, allowing accurate quantitation from all data points. From the 200 x 200 raster scan pattern, individual B-scans show that typical drusen elevate the RPE. The segmentation algorithm produces an RPE segmentation map that reveals the contours and elevations of the drusen. The 3-D view resembles a mountain range (Figure 1).

Another algorithm creates a model of a normal RPE without drusen. This virtual RPE floor is mathematically subtracted from the RPE segmentation map, providing an RPE difference map, allowing extraction of both area and volume measurements. This map can be fit over a color fundus image, demonstrating the correlation between the 2 imaging strategies. Drusen measurement reproducibility was established in a study assessing 103 eyes from 74 patients, with 5 SD-OCT scans of each eye made at the same visit.3

**Drusen**

In a 2-year study of 143 eyes, several patterns of drusen growth were observed.4 After 12 months, drusen volume increased in 48% of eyes, remained stable in 40%, and decreased in 12%. Clinical experience confirms that increasing drusen size indicates a poor prognosis, and coalescing drusen can produce larger drusenoid PEDs over 12 months. Three outcomes can accompany a decrease in drusen size: 1) no significant anatomical abnormality identified, 2) formation of choroidal neovascularization, or 3) formation of GA. Although drusen can appear almost completely resolved without any obvious sequelae, the resolution of drusen can also precede the accumulation of fluid under the retina, and this leakage represents new choroidal neovascularization. Alternatively, drusen can progress to GA, the more serious of the 2 pathologic outcomes. Neovascularization can be treated, but no treatment is available for GA. This progression can be seen on autofluorescence as an area of hypofluorescence, on the B-scan as increased light penetration into the choroid, and on the *en face* drusen map as an area of increased brightness where the drusen have disappeared (Figure 2).

Additional longitudinal information was obtained from a natural history study that followed 16 eyes of 11 patients with drusenoid retinal PED secondary to AMD.5 SD-OCT was used to monitor changes in drusenoid PED area and volume and development of advanced AMD.
B-scan showing cystoid degeneration in the retina overlying a PED indicated an unfavorable prognosis. The RPE breakdown was visible on the B-scan as increased light streaking into the PED and the choroid, indicating decreased reflectance of the RPE. As disease progressed, increased penetration under the RPE was observed, with increased cystoid degeneration of the retina, followed by the contents of the PED exuding through the break in the RPE into the retina. Probable activation of microglia and phagocytic cells followed, represented as the hyporeflective foci observed around the exuded material. With continued penetration, the retina collapsed onto the exposed Bruch’s membrane, with loss of photoreceptors and formation of GA and loss of the outer retinal structures. This process occurred in 5 (31%) of the eyes in this study.

**Geographic Atrophy**

Historically, color fundus and autofluorescence imaging have been the mainstays for following progression of GA. Hypofluorescence associated with the loss of RPE provides a more obvious indication of the enlargement compared with color fundus imaging. Fluorescein angiography has also been used to identify and quantify GA. SD-OCT has become an extremely useful modality for identifying this form of AMD, providing an OCT fundus image. The same 200 x 200 raster A-scans are used for the Cirrus HD-OCT fundus scanning pattern, which acquires 40,000 A-scans in <2 seconds. The fundus image is obtained from the collection of the B-scans into a 3-dimensional data set, summing the signal of each A-scan and displaying their values en face. GA is represented as a bright area of increased light penetration into the choroid where the RPE and choriocapillaris are absent. When intact, these structures cause light to scatter before it can penetrate the choroid. With autofluorescence, GA appears as a dark area, but the configuration of GA appears the same so both imaging strategies can be used interchangeably. The visualization of GA using SD-OCT was improved by using the sub-RPE “slab,” or enhanced OCT fundus image. Using the same scan pattern, this modified imaging strategy is based only on the light reflected from under the RPE. The area affected by GA is brighter, with greater contrast, distinguishing the areas of GA more precisely. An algorithm allows automated segmentation, with area measurements showing extremely good agreement with manual measurements.

Patients with AMD treated with an anti-VEGF agent may have an increased risk of progressing to GA. SD-OCT fundus images can allow visualization of this process concomitant with other changing morphologic features. This modality can also follow other changes that occur during...
GA progression. Electrophysiologic and microperimetric testing and histopathology studies have shown that photoreceptor atrophy can occur at a distance from the edge of the atrophy. The use of SD-OCT imaging of the outer retina to predict progression of GA has been explored.

A prospective study followed 30 eyes with GA secondary to AMD, with a baseline area between 1.8 and 18 mm², for at least 1 year. The same scan pattern was used as for the other investigations; and a different algorithm was used to extract the desired information. A 20-μm slab containing the outer retinal IS/OS/E (inner segment/outer segment/ellipsoid zone) boundary provides the en face image. In normal eyes, with the exception of shadows from the large retinal blood vessels, the macula outside the foveal center is uniformly bright, with a slightly darker foveal center due to elevation of the inner segment/outer segment/ellipsoid (IS/OS/E) region outside the slab.

Longitudinally obtained en face images superimposed with a whitened image of baseline GA from the corresponding sub-RPE slabs show the darkened area around the GA that eventually progresses to GA. The growth of GA into the dark area is shown by the progressing white line over the course of 1 year (Figure 4). B-scans through these dark areas compared with the locations on the corresponding en face images allow correlation between the dark areas and the associated pathology. In the larger dark areas, the outer retina is disrupted, photoreceptors are lost, the outer nuclear layer is thinner, the photoreceptor layers are gone, and the IS/OS/E is significantly disrupted. Atrophy is apparent in the area of GA, and the bordering dark areas represent photoreceptor disruption.

In some cases, the focal dark baseline areas predicted the asymmetric direction of GA progression. Well-demarcated dark areas adjacent to GA were classified as focal. Bilateral symmetry was observed in the pattern of outer retinal disruption extending beyond the GA border, accurately predicting GA progression over 1 year in 13 of 30 eyes. The other eyes had a disruption area that was much larger than the progression area and was referred to as a diffuse pattern.

Some GA cases follow a diffuse pattern of growth, exhibiting a poorly demarcated dark area. These cases are usually associated with reticular pseudodrusen, characterized by a lacy, ribbon-like pattern shown on autofluorescence. When the en face IS/OS/E slab image is superimposed, the associations of the ribbon-like structures with the disruptions in the IS/OS/E band are visible. The lesion spreads diffusely into the area of reticular pseudodrusen. By noting these characteristics, in addition to imaging GA, users can identify reticular pseudodrusen and predict the growth characteristics of the GA.

IS/OS/E slab imaging around the GA shows outer photoreceptor disruption up to thousands of microns distant from the edge of the atrophy. Outer photoreceptor disruption that precedes GA can predict its appearance in some patients.

Vascularized PEDs

Patients with a vascularized PED involving the fovea were not included in the pivotal anti-VEGF trials. The response to anti-VEGF in eyes with a PED plus subretinal fluid can now be monitored using OCT. Resolving subretinal fluid followed by resolution of the PED can be visualized using retinal thickness and RPE maps to supplement the scanned images. Similar to drusen, vascularized PEDs are elevations of the RPE for which area and volume can be measured using an SD-OCT RPE elevation algorithm. Qualitative and quantitative assessments of RPE using this automated algorithm are highly reproducible.

SD-OCT imaging strategies for the management of PEDs include the retinal thickness map that shows the
subretinal and intraretinal fluid, the RPE map showing the PED, and PED volume map data that can be used to follow patients on anti-VEGF therapy. Patients with a PED can be treated to minimize its volume, similar to following macular fluid in and under the retina. The ability to make multiple follow-up investigations allows for accurate monitoring of PED volume increases and decreases, as the dosing interval is varied and the PED moves toward resolution.

Scans from 14 eyes with vascularized PEDs that were undergoing PRN treatment with anti-VEGF agents were assessed retrospectively using the algorithm to measure the area and volume of PEDs. Treatment was withheld at 57 visits; however, PED volume was increased at 8 of these visits. The decision to observe in those 8 patients was based on intraretinal and subretinal fluid status. Macular fluid, an indication for injection, reappeared at the next visit, but the increased PED volume at the previous visit predicted the appearance of fluid at the subsequent visit. Monitoring changes in PED volume may be used as an additional criterion in eyes undergoing SD-OCT–guided anti-VEGF therapy, allowing treatment before subretinal and intraretinal fluid reaccumulates in the macula.

Available SD-OCT Instruments

Fourier transformation, which provides the mathematical basis for SD-OCT technology, is nonproprietary, and 7 SD-OCT systems developed from the operation are currently available. The systems are distinguished by their advanced features and specific analysis protocols. Variations in standard features among products include axial resolution ranging from 4 to 7 μm and A-scans per second from 18,000 to 40,000. Some instruments have fixed scanning patterns, while others offer a range of choices. Image management, processing times, and output formats in some devices may lead to underuse of advanced features.

Lack of standardization can contribute to varying results among devices. While the Cirrus HD-OCT uses the RPE in its segmentation algorithm, the Spectralis uses Bruch’s membrane. When patients with AMD were examined using 6 devices, mean retinal thickness ranged from 189 to 384 μm among the 6 instruments. These differences can complicate comparisons among studies and case discussions among practitioners, and standardization of this technology has been advocated. In addition, artifact incidences among instruments have been reported. These important data can contribute to the continuing improvements and upgrades in software and algorithms that are rapidly being made in this dynamic field.

References


Case 1: Hemorrhagic PED on Anti-VEGF Therapy

A 65-year-old woman enrolled in a clinical trial for dry macular degeneration in the fellow eye was receiving biweekly follow-up according to the study protocol. The frequent follow-up allowed for detailed OCT analysis of the eye receiving anti-VEGF treatments as needed for vascularized, hemorrhagic PED (Figure 1A). Four weeks after the second injection, her subretinal fluid had resolved, and the volume of the PED had decreased; she was then managed by observation (Figure 1B). At her next 2-week visit, the PED volume had increased; however, no fluid was apparent under or in the retina, and observation was continued. Eight weeks following her second injection, a significant increase of fluid was observed on both the B-scan and the RPE map, with a significantly increased PED volume. A third anti-VEGF injection was given. Four weeks after the third injections, the patient again had resolved fluid and a decreased PED volume.
Case 2: The Secret Life of Drusen Revealed by SD-OCT Imaging

A 61-year-old woman was followed over 3 months with fundus autofluorescence and OCT. The color fundus images during that interval showed little difference; however, the drusen maps clearly showed an increase in volume and area (Figure 2A), with vision decreasing from 20/25 to 20/30. These drusen spontaneously resolved and were undetectable at 6 months, and vision returned to 20/20 with no recurrence after years of follow-up.

The fellow eye had initial visual acuity of 20/25, which decreased to 20/30 at 9 months. Drusen in this eye increased over 3 months, then began to resolve at 6 months, with almost complete resolution by 9 months (Figure 2B). Little change was detectable in color fundus images from these 4 time points. Therefore, color fundus imaging cannot give a precise indication of status of and changes in drusen.
Diabetic retinopathy, primarily in the form of diabetic macular edema (DME), is the leading cause of blindness in US residents younger than 65 years. The 1985 Early Treatment Diabetic Retinopathy Study (ETDRS) established clinically significant macular edema as a clinical diagnosis. Whether laser treatment was needed or not was decided by looking into the patient’s eye, basing the diagnosis on how far from the center of vision thickening or exudate occurred. That definition was used for many years, and laser was the only available treatment.

Current investigations include fluorescein angiography, with optical coherence tomography (OCT) increasing in importance for determining when treatment is necessary and for monitoring treatment response. Fluorescein angiography provides information that cannot be obtained with OCT, which cannot detect macular ischemia or determine specific blood vessels that are leaking. Angiography results continue to provide the basis for laser treatment.

Historically, an extremely small region of the retina was observed with fluorescein angiography. The ETDRS study examined a larger region, referred to as the 7 standard fields (7SF), which are still used. However, ultra wide-field fluorescein (UWFA) angiography devices allow observation far into the periphery, showing 3.2 times more retinal surface area than the 7SF. In a study of 218 eyes in patients with diabetes, UWFA showed 3.9 times more nonperfusion ($P<0.001$), 1.9 times more neovascularization ($P=0.036$), and 3.8 times more panretinal photocoagulation ($P=0.001$) compared with 7SF. Retinal pathology that was not evident in a 7SF image was demonstrated in 10% of the eyes. Although still in its infancy for imaging patients with diabetes, the improved retinal visualization possible with UWFA may contribute to better evaluation and management of patients with diabetic retinopathy.

Imaging DME

Leakage pattern guides treatment for patients with diabetic retinopathy. Areas of focal leakage, showing retinal thickening surrounding clusters of microaneurysms, indicate that a patient may benefit from laser treatment in addition to anti-VEGF injection. Diffuse leakage, with obvious macular cysts and abundant exudates, has a less obvious source. Although laser treatment was used in the past, anti-VEGF and steroid medications may be more beneficial in cases with diffuse leakage. However, the degree of leakage on fluorescein angiography does not correlate with visual acuity or clinical outcomes and does not illustrate vitreoretinal abnormalities. Therefore, OCT has become vital in the diagnosis and management of these patients.

OCT can produce thickness maps and line scans. Cube scan protocols give a 3-D view of the entire retina and illustrate retinal thickness and other abnormalities. Retinal thickness maps indicate areas of leakage, which correlate well with clinical leakage, fluorescein leakage, and decreased vision. Some retinal thickness map applications allow registering thickness changes over time, facilitating the evaluation of treatment effectiveness. The colored maps are always of interest to patients and can be used to help explain treatment decisions.

However, retinal maps do not provide all the necessary decision-making information, especially in the early treatment stages. A line scan (B-scan), particularly with averaging, has become an “optical biopsy” of the retina. Line scans provide structural details and vitreomacular interactions, demonstrating distinct patterns of DME that may have both prognostic and management indications.

Leakage patterns are not mutually independent but can occur together in the same eye. The most commonly observed is a sponge-like DME, which has been reported in 55% to 96% of cases. Fluid leaks from the retinal vasculature into the outer retinal layers and absorption of the exudate resemble a sponge, which often compresses the inner retinal layers. This morphology, with increased thickening, correlates well with reduced visual acuity. Sponge-like swelling that occurs alone responds well to laser treatment.

Diabetic cystoid macular edema (CME) is observed in 30% to 57% of cases. The bubble-shaped cystoid changes are more damaging than the sponge-like DME. The growing cysts coalesce, exerting a severe detrimental effect on visual acuity. Electroretinogram testing reveals a greater reduction in macular sensitivity compared...
with sponge-like swelling. Laser treatment is usually ineffective for these patients. Treatment includes steroids (triamcinolone, dexamethasone implant) and anti-VEGF agents (0.3 mg ranibizumab, bevacizumab, or aflibercept off-label). Surgery has not been proven effective but may be considered in recalcitrant cases.

A third leakage pattern, serous retinal detachment without posterior hyaloid traction, has been reported in up to 15% of patients. The detachment occurs in the fovea, thereby elevating it.

A unique pattern, occurring in 16% of patients in 1 study, is DME due to posterior hyaloid traction. Traction is often missed on examination, especially when the hyaloid is only slightly detached from the retinal surface. A smaller subset of patients, reported to occur in 2% to 3% of patients, also has a traction retinal detachment. Surgery is the standard treatment in these patients. Intravitreal injections and laser treatment are ineffective. Recognizing this pattern early in its development is important to allow performing surgery as soon as possible, rather than delaying proper treatment by using ineffective medications.

Posterior hyaloid traction without a traction retinal detachment was reported in 14% of cases. Laser is usually ineffective. Although these patients may respond to medical therapy, the swelling recurs quickly, and the practitioner must decide whether to continue with injections or perform surgery, which may improve macular edema and visual acuity yet carries the risks of an invasive procedure.

In summary, at least 6 different morphologic patterns of DME can be identified using OCT. These represent distinct entities that may require individual treatment regimens. Identifying these patterns may allow more effective management of these patients.

### Retinal Vein Occlusion

Retinal vein occlusions (RVOs) are the second most common type of retinal vascular disease and can occur in a branch of the retinal vessels (BRVO), in the central retinal vein (CRVO), or in 1 trunk of the central retinal vein (hemi RVO). The incidence of RVO in the United States is approximately 180,000 eyes per year, of which BRVO accounts for nearly 80%. The 3 types of RVO look similar on OCT, which cannot be used alone for diagnosis. Angiography must be done at baseline to verify that the leakage shown on OCT is due to occlusion of the retinal vein and to differentiate among the RVO forms.

After the initial diagnosis, OCT is valuable for following these patients and monitoring their response to treatment. However, subgroup analysis of morphologic characteristics does not have prognostic value. Laser therapy is not effective in CRVO and in the other forms is not as effective as anti-VEGF therapy. Therefore, intravitreal injections are the mainstay therapy for RVO. A change analysis can help guide decisions to provide further injections. If patients are not realizing their desired visual gain, showing them the positive effects treatment is having on the retina can facilitate a decision to continue treatment until vision improves.

### Ischemic Retinal Vein Occlusions

During diagnosis, differentiation between ischemic and nonischemic RVO is important and can be done with fluorescein angiography. Ischemic RVO, which is more likely to progress to neovascular complications, is defined as having >10 disc areas of peripheral nonperfusion. Severe vision loss is common, with rubeosis developing in 70% of patients and neovascular glaucoma in 50%. Ischemic RVO requires careful treatment decisions and frequent follow-up; panretinal photocoagulation reduces complications but also ablates the peripheral visual field.

Patients with extensive nonperfusion with neovascularization benefit from early laser treatment with diligent follow-up to ensure neovascular glaucoma does not develop. Patients without neovascularization should also be followed closely, but do not need laser treatment.

Both OCT and fluorescein angiography assist with decisions about laser treatment and follow-up frequency. In 1 report, 69% of eyes with foveal thickness \( \geq 700 \mu m \) had peripheral ischemia. Targeted panretinal photocoagulation to the areas of nonperfusion may be warranted when OCT retinal thickness map images reflect a worsening condition after several anti-VEGF injections, and wide-field angiography reveals extensive nonperfusion in the periphery. Continuing the injections is warranted, as treating the areas of nonperfusion may decrease the VEGF drive and allow improved outcomes following the injections, leading to their discontinuance.

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**Table 1. DME Morphologic OCT Characteristics**

<table>
<thead>
<tr>
<th>Prevalence in Study</th>
<th>Sponge-like DME</th>
<th>Diabetic CME</th>
<th>Serous RD no PHT</th>
<th>DME due to PHT</th>
<th>With traction RD</th>
<th>No traction RD</th>
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<td>Source</td>
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KEY: CME, cystoid macular edema; DME, diabetic macular edema; PHT, posterior hyaloid traction; RD, retinal detachment.

Choroidal thickness can be measured using OCT in patients with RVO. In a study of 36 eyes, choroidal thickness was significantly greater in eyes with CRVO compared with the fellow eye \((P<0.01)\).\(^{22}\) Eyes treated with anti-VEGF medications experienced significant decreases in choroidal thickness \((P<0.01)\). VEGF increases vessel dilation and capillary permeability, particularly in the choroid, and is increased in patients with CRVO. The inner retinal ischemia from CRVO increases choroidal blood flow to better supply the watershed area between the inner and outer retina.

Another common OCT finding in RVO is posterior vitreous attachment to and putting traction on the retina.\(^{23}\) Patients with CRVO were more likely to have attached posterior vitreous cortex compared with patients with BRVO, and attachment in both forms of RVO was considerably more prevalent compared with age-matched historical controls.

Summary

Patients with diabetes have many morphologic characteristics that have prognostic and management significance. Patients must be classified at baseline to determine the best management course. Morphologic characteristics in RVO do not carry the same prognostic significance; however, as with diabetic retinopathy, OCT alone cannot be used to manage these patients. Fluorescein angiography must also be used to determine prognosis and the necessary follow-up frequency. In the era of retinal pharmacotherapy, OCT plays a critical role in treatment decisions and allows detection and monitoring of subtle cases of macular edema. OCT can detect concomitant vitreo-retinal interface pathology (eg, epiretinal membrane), retinal thinning, or loss of the ellipsoid zone in the outer retina, which may explain persistent vision loss. Finally, OCT provides an engaging platform for improved patient education.

References


Case Studies

Michael S. Ip, MD

Case 1: Visual Compromise with Diffuse Edema

A 58-year-old woman was referred for further management of DME in the right eye. She had been treated twice with focal laser with a visual acuity of 20/60 and 20/50 in the left eye. Her right eye exam was remarkable for diffuse macular edema with hard exudate in a nearly circuate pattern (Figure 1A). Fluorescein angiography showed a lack of significant ischemia or leakage in the central foveal region. Her baseline OCT showed significant macular edema on the axial scan, with significant diffuse macular edema visible on both the topographic and ETDRS maps.

The patient was treated with 1 injection of bevacizumab, with subsequent OCT images showing significant resolution of the macular edema (Figure 1B). However, her visual acuity remained at 20/60 after 3 injections despite OCT showing her edema was almost completely resolved on both axial scans and maps (Figure 1C). Injections were extended, and she was observed based on lack of visual improvement. Without OCT, the resolution of the edema would have been difficult to observe, and injections may have been continued based on her vision deficit.

Case 2: Following a Patient with Good Vision and Mild Diabetic Macular Edema

A 74-year-old man with good vision and no significant visual complaints was followed for DME in both eyes. His only interventions have been successful systemic management of blood sugar, blood pressure, and lipids. OCT scans revealed absence of macular edema in the central subfield, with mild macular edema surrounding the central subfield (Figure 2A).
Six months later the macular edema had shifted, with varying amounts in the inner and outer subfields compared with the prior scan (Figure 2B). No macular edema appeared in the central subfield, and the axial scan was not significantly changed.

One year from baseline, the edema had increased in the inner and outer subfields (Figure 2C). Importantly, macular edema was present in the central subfield, and axial scans showed no significant change.

This case demonstrates that OCT can aid in detecting mild macular edema and observing the condition over time to monitor pattern shifts. Although the inner and outer subfields changed, the central subfield remained stable; therefore, no intervention was required. Using OCT, deterioration of this patient’s status might be detected before he becomes symptomatic. This provides an excellent tool to educate patients about the nature of their disease and can help provide motivation when better systemic control is needed.

**Case 3: Foveal Monocyst**

A 63-year-old man was referred with DME and a history of CRVO in his right eye. He had a previous radial optic neurotomy, resulting in poor visual acuity in his right eye. He reported reduced vision and metamorphopsia in his better left eye. His vision was 20/100 and 20/40 in his right and left eyes.

Initial examination of the left eye revealed mild non-proliferative disease with a foveal monocyst on OCT, supporting his report of reduced visual acuity (Figure 3A). This condition is difficult to detect on the color fundus photograph. Topographic and ETDRS maps show that most of his edema was in the central subfield, without any significant edema in the inner and outer subfields.

The patient was treated with bevacizumab. His symptoms improved, and visual acuity increased to 20/30; however, he was not satisfied, so the injections were continued with follow-up at 4-week intervals. His visual acuity remained at 20/30 despite 4 treatments, and the cyst did not resolve (Figure 3B). The patient was
switched to ranibizumab injections, and visual acuity improved to 20/20 after the first injection, which was accompanied by anatomic improvement on OCT. He continued on monthly injections. When the central foveal cyst was almost completely resolved after the 14th injection (Figure 3C) with stable visual acuity, he was extended to a 6-week injection interval.

In this case, OCT was useful in correlating the initial symptoms with the retinal anatomy. The axial scans showed subtle effect differences between 2 anti-VEGF drugs, with ultimate resolution of the foveal cyst.

**Case 4: BRVO with Macular Edema**

An 82-year-old woman was referred with BRVO in the right eye and 20/400 visual acuity. Hemorrhages were visible on the color fundus photograph, and leakage and areas of ischemia were noted on fluorescein angiography (Figure 4A). OCT revealed extensive macular edema with considerable disruption of the normal foveal architecture, and significant cysts and thickening of the retina were apparent.

The patient was treated with bevacizumab, and after 11 injections her visual acuity had increased to 20/40. The residual visual reduction may be due to damage from the prior edema; however, a subtle epiretinal membrane is apparent on the surface of the retina, which may account for the reduced vision. Her injection interval was increased from 6 to 8 weeks; however, at the next follow-up, after the 12th injection, vision had not changed although the OCT showed some worsening (Figure 4B). Based on OCT findings, the patient returned to the 6-week injection interval, with subsequent improvement in OCT findings (Figure 4C), and vision improving to 20/30.
1. Normal posterior vitreous detachment:
   A. Can be seen as early as 4 years of age.
   B. Is rarely observed at less than 60 years of age.
   C. Is characterized by persistent hyaloid attachment at the macula with traction.
   D. Occurs when an imbalance develops between liquefaction and weakening of the vitreoretinal adhesion.

2. Disorders associated with persistent hyaloid attachment at the macula with traction include:
   A. Epiretinal membrane (ERM) and lamellar macular hole (LMH).
   B. LMH and full-thickness macular hole (FTMH).
   C. ERM and vitreomacular traction (VMT).
   D. VMT and FTMH.

3. A 58-year-old woman presents with a lamellar macular hole and reduced vision (20/60). An appropriate management plan includes:
   A. Immediate vitrectomy to improve her vision.
   B. Observation, as surgery is rarely effective for hole closure in these patients.
   C. Observation, with vitrectomy an option if follow-up reveals deterioration in her vision or retinal anatomy.
   D. Immediate vitrectomy only if SD-OCT-defined characteristics are predictive of success.

4. Which is an appropriate management plan for a 52-year-old man with a phakic left eye with a 400-µm full-thickness macular hole with VMT?
   A. Immediate vitrectomy because ocirplasmin is not effective in holes ≥ 400 µm.
   B. Initial treatment with ocirplasmin for 3 months followed by vitrectomy if closure is not achieved.
   C. Watch and wait, with surgery indicated if the hole expands.
   D. A single treatment with ocirplasmin, with vitrectomy an option if closure does not occur within 28 days.

5. Geographic atrophy:
   A. Appears on an OCT B-scan as penetration of light into the choroid.
   B. Typically accompanies an increase in drusen size.
   C. Is less difficult to manage than choroidal neovascularization.
   D. Cannot be monitored using fluorescein angiography.

6. A patient with a vascularized pigment epithelial detachment (PED) is undergoing OCT-guided anti-VEGF treatment.
   A. Retreatment should be based only on reappearance of macular fluid pending studies on PED volume correlation.
   B. PED volume increase may predict an increase in macular fluid, which may allow treatment before macular fluid reaccumulates.
   C. Anti-VEGF pivotal trial data failed to show a correlation between PED volume increase and macular fluid reappearance.
   D. Optimal treatment can be based on assessment of retinal thickness maps.

7. The most A-scans per second on commercial SD-OCT instruments is:
   A. 5,000
   B. 10,000
   C. 20,000
   D. 40,000

8. In patients with diabetic macular edema (DME):
   A. OCT has become a standalone diagnostic and management tool.
   B. Fluorescein angiography is necessary because OCT cannot detect macular ischemia or determine which blood vessels are leaking.
   C. OCT results can provide the basis for laser treatment.
   D. Fluorescein angiography is necessary because OCT cannot detect posterior vitreous detachment or indicate specific areas of leakage.

9. OCT imaging in a patient with DME and unchanged visual acuity referred after 3 anti-VEGF injections:
   A. Is unnecessary because the injections should be continued based on her visual defect.
   B. Could suggest injections can be extended if edema resolution is demonstrated.
   C. May indicate laser treatment is warranted if diffuse rather than focal leakage is shown.
   D. Can provide sufficient treatment decision information from retinal map assessment alone.

10. Laser treatment is often effective in:
    A. Diabetic cystoid macular edema.
    B. DME due to posterior hyaloid traction with traction retinal detachment.
    C. DME due to posterior hyaloid traction without traction retinal detachment.
    D. Sponge-like DME.
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Advanced Optical Coherence Tomography

Applications for the Retina Specialist

POSTTEST

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*Time spent on this activity: Hours ☐ Minutes ☐
(reading articles and completing the learning assessment and evaluation)
This information MUST be completed in order for the quiz to be scored.

THE MONOGRAPH AND TEST EXPIRE AUGUST 1, 2015

PRINT OR TYPE

Last Name        First Name        Degree

Mailing Address

City            State            Zip Code

Date of Birth (used for tracking credits ONLY)

Phone Number        FAX Number        E-mail

EVALUATION (must be completed for your CME Quiz to be scored)

Your evaluation of this activity is extremely important as it allows for us to plan for future educational programs. Please take a moment to answer the following questions, filling in circles completely and writing answers legibly.

1. Overall the activity supported achievement of the identified learning objectives. Yes No
2. This activity better prepared me to care for my patients. Yes No
3. The content covered was useful and relevant to my practice. Yes No
4. The activity was presented objectively and was free of commercial bias.* Yes No
5. Future activities concerning this subject matter are necessary. Yes No
6. The activity addressed and provided strategies for overcoming barriers to optimal patient care. Yes No
7. The activity reinforced my current practice patterns. Yes No

*If you indicated that the activity was not free of commercial bias, please provide additional comments here:

________________________________________________________________________
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8. What educational topics would be of value to you, for future CME activities? Please be specific.

________________________________________________________________________

9. Approximately what percentage of the activity’s content was NEW to you? 

☐ 0%         ☐ 25%         ☐ 50%         ☐ 75%         ☐ 100%

10. Do you believe this program: Y=Yes N=No 1=N/A

Increased your knowledge about the subject matter? Y N 1
Will improve patient outcomes in your practice? Y N 1
Helped you to have a better understanding of the topics? Y N 1
Gave you a new perspective on the information which may help to improve your practice? Y N 1
Provided you with resources to use in your practice and/or with your patients? Y N 1

11. Planned Changes to Practice - Please respond to the statements below using the following scale: Y=Yes N=No 2=I Already Do This in My Practice 1=N/A

I plan to make the following changes to my practice.

- Revise use of OCT in managing patients with AMD
- Revise criteria for managing patients with disorders of the vitreomacular interface
- Implement new imaging criteria for patients with macular edema

Other (Please provide below):__________________________

12. Barriers to Practice - These are the barriers I face in my current practice setting that may impact patient outcomes: (Select all that apply)

☐ Lack of evidence-based guidelines
☐ Changing standards of care and evidence-based guidelines
☐ Lack of time to spend with my patients
☐ Organizational/institutional limitations
☐ Cost of therapies and reimbursement issues
☐ Patient adherence/compliance issues
☐ Treatment-related adverse events
☐ Contraindications to therapies
☐ Resistance to therapy
☐ Difficulty in keeping up with evolving evidence to make treatment decisions
☐ Coordination of care with other care providers
☐ Other (Please provide below):__________________________

13. How confident are you in using OCT to diagnose and guide management of macular disorders?

☐ Extremely Confident
☐ Very Confident
☐ Somewhat Confident
☐ Not at All Confident

14. Approximately how many patients do you assist per week using optical coherence tomography? 

☐ Less than 10
☐ 10 to 30
☐ 31 to 50
☐ More than 50
☐ N/A

15. Please indicate your degree. (Please select one only.)

☐ MD/DO
☐ PhD
☐ NP
☐ RN/BSN/MSN
☐ RPh/PharmD

16. Please indicate your specialty (Please select one only.)

☐ Ophthalmology
☐ Cornea/External Disease
☐ Retina/Vitreous
☐ Other__________________________

17. How many years have you been in practice?

☐ 0-5 years
☐ 6-15 years
☐ 16-25 years
☐ 26-30 years
☐ 31+ years

18. Would you recommend this activity to your peers? Yes No

CME ACTIVITY REQUEST

☐ Yes, I would like the opportunity to earn CME credits through activities sponsored by Vindico Medical Education.

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